Inducibility at EPS was predicted with greatest specificity by the presence of RA in early repolarization (T_{early} Specificity = 0.88), compared to RA involving later components of the T wave; T_{mid} (0.79), T_{late} (0.63), or the ST segment (0.71). Similarly, RA involving early repolarization was the most specific marker for reduced arrhythmia-free survival (Specificity's: T_{early} 0.87, T_{mid} 0.83, T_{late} 0.58, ST 0.80).

Conclusions; RA of the terminal component of the T wave is present in most patients and is not necessarily a specific marker for arrhythmia vulnerability. In contrast, RA involving the early phases of repolarization (T_{early}) appears to have the most prognostic significance. RA related arrhythmogenesis may be due to an underlying disturbance affecting early repolarization.

926-26 Electrophysiologic Testing, Electrical Alternans and Signal Averaged Electrocardiography as Predictors of Arrhythmia-free Survival

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Arrhythmia-free survival was analyzed retrospectively in 47 patients who underwent programmed electrophysiologic (EP) testing as well as electrical alternans (EA) and signal averaged electrocardiography (SAECG) measurements.

We compared the accuracy of (i) electrophysiologic testing (inducible ventricular tachycardia or fibrillation), (ii) electrical alternans (alternans ratio > 3), (iii) signal averaged ECG (QRS duration > 114 msec or LAS > 38 msec or RMS 40 < 20 μV) and (iv) the combined use of EA and SAECG (a patient was classified positive if EA was positive and SAECG was positive or indeterminate) to predict the arrhythmia-free survival of these patients. SAECG was deemed indeterminate if the QRS duration of any of the unfiltered Frank leads was greater than 120 msec (n = 11). The accuracy of predicting arrhythmia-free survival was estimated by computing actuarial arrhythmia free-survival at 20 months and comparing 20 month survival rates with EP, EA and SAECG testing. The results are:

	Sensitivity	Specificity	PV+	PV−	RR	Accuracy	P
EP	71%	78%	36%	94%	5.89	77%	0.0090
EA	89%	82%	53%	97%	17.06	83%	<0.0001
SAECG	50%	72%	18%	92%	2.27	69%	0.3706
EA & SAECG	88%	91%	68%	97%	24.61	90%	<0.0001

PV+: positive predictive value, PV−: negative predictive value, RR: relative risk

Conclusions; EP, EA and EA & SAECG were significant predictors of arrhythmia free survival; SAECG alone was not a significant predictor. In this patient population, EA or EA combined with SAECG provided a powerful measure of risk comparable or superior to EP.

926-27 Probucol-associated Arrhythmic Events: Further Evidence for Increased Female Predisposition to Acquired Long QT Syndromes

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Recent data suggest an increased female susceptibility to torsade de pointes (TdP) during exposure to cardiac drugs that prolong the QT interval. We investigated possible gender differences in the occurrence of arrhythmic events in pts taking Probucol (P), a lipid lowering agent that prolongs QTc. A MEDLINE search along with data obtained from the Food and Drug Administration, identified a total of 16 reported pts with arrhythmic events (TdP-10, VF-2, VT-2, cardiac arrest-2) during P therapy (median P dose 1000 mg/day, for median duration 1 yr). Of these 16 pts, 15 (94%) were female; yet, during approximately the same time period of these reports (from 1979–1991), nationwide P exposure, according to a large pharmacological database (IMS America), was only 60% female ($p < 0.01$). Among 10 of the reported pts having known QT interval data, 8 (80%) exhibited marked QTc prolongation (>0.60sec) on P. Nine of the 16 pts had additional potential causes of QT prolongation (Class IA agents-3, hypokalemia-2, prolonged baseline QTc-2, prolonged baseline QT/hypokalemia-1, Class IA agent/hypokalemia-1). In the 3 reported cases of TdP *without* concomitant causes of QT prolongation, all were female. From pooled studies, we also analyzed a total of 357 asymptomatic pts (64% female) with *normal* baseline QTc (≤0.44 sec) in whom QTc intervals were obtained both prior to and during P therapy (median P dose 500 mg/day, for both men and women). On P, QTc was >0.45 sec in 16% of women (up to 0.51 sec) vs 3% men (up to 0.49 sec) [$p < 0.001$] and was ≥0.47 sec in 8% of women vs 2% men [$p < 0.03$]; logistic regression revealed pre-P QTc ($p = 0.0001$) and female gender ($p = 0.01$) to be significant predictors of P-induced QTc prolongation (>0.45 sec).

Conclusions; (1) Women appear to have increased susceptibility to Probucol-associated arrhythmic events (primarily TdP); (2) Probucol-associated arrhythmic events occur mainly, but not exclusively, in pts with additional conditions which may prolong QTc; (3) Even in the absence of TdP, women are more likely to exhibit significant Probucol-induced QTc prolongation, suggesting a clinical continuum in the differential QT interval response of women to the drug.

926-28 Ventricular Tachycardia After Inferior Wall Myocardial Infarction: Predominance of Basal Locations for Critical Slow Conduction Zones

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Inter-patient similarities in coronary and ventricular anatomy may cause reentry circuit slow conduction zones (SCZ) to form in certain locations after myocardial infarction (MI). We assessed the location of reentry circuit SCZs identified using entrainment techniques during catheter mapping and ablation in 14 patients with an old inferior wall MI. The inferior wall was divided into 9 regions. Of 34 different ventricular tachycardias (VT) having a mean cycle length of 408 ± 82 msec, SCZs were identified in 21 (62%). At 18 of these 21 sites (86%), the SCZ was in a basal region near the mitral annulus ($p < 0.01$ vs all other regions). In 6 VTs, the SCZ extended into two regions.

	IN-SEP	INF	INF-LAT	Total (27)
BASAL	3	13	6	22
MID	1	2	0	3
APEX	0	2	0	2

SEP = septum, INF = inferior, LAT = lateral

Conclusion; The prevalence of SCZs near the base after inferior MIs suggest a possible role of the mitral annulus in defining the margins of the reentry circuit.

926-29 Mechanisms for Pacing-induced Slowing in Conduction within the Reentrant Ventricular Tachycardia Circuit. Importance of True Fatigue in Conduction

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Background; It has been demonstrated experimentally and suggested clinically that post-repolarization refractoriness can produce pacing-induced conduction delays in ventricular tachycardia (VT) circuits.

Material and Methods; To evaluate the contribution of true fatigue in conduction to such phenomena during burst pacing, we compared the response to single extrastimuli entering the reentrant circuit with a known coupling interval (CI) to the response to synchronized 15 to 21 beat bursts with repetitive fast penetration of the VT circuit. Each extrastimulus was preceded by a 15 to 21-beat "slow" pacing train producing entrainment of the VT to ensure that the extrastimuli entered the VT circuit with an interval similar to the CI at the pacing site. The degree of fatigue was quantitated by subtracting the first post-pacing interval (FPPI) after each extrastimulus from the FPPI after a pacing burst of a cycle length (CL) identical to the CI of that extrastimulus. We evaluated the degree of fatigue during 44 CL in 11 VT (mean VT CL 340 ± 47 ms).

Results; FPPI was shorter after extrastimuli (465 ± 79 ms) than after their correspondent bursts (486 ± 93 ms, $p < 0.001$ by paired t-test). The degree of fatigue was related to the pacing CL (29 ± 42 ms for bursts CL <85% of VT CL vs 11 ± 21 for bursts CL ≥ 85% of VT CL, $p < 0.05$). There was a trend towards a higher degree of fatigue in VT while on antiarrhythmic drugs (25 ± 41 ms vs 11 ± 9 ms, $p < 0.1$).

Conclusion; True fatigue in conduction adds to the possible conduction delays related to refractoriness within clinical reentrant VT circuits. This may explain why, not only rate but duration of pacing, can be critical for VT termination, particularly with fast pacing rates.

926-30 Cycle Lengths of Ventricular Tachycardia Recurrences in Patients with Coronary Artery Disease: Relationship to Clinical Presentation and Induced Tachycardias

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Using stored electrograms, we analyzed the cycle lengths of spontaneous ventricular tachycardia (VT) recurrences (RCL) after implantation of a Ventritex Cadence™ device in 22 patients with remote myocardial infarction whose initial presentation and followup were on no antiarrhythmic drug therapy. The initial clinical presentation prior to Cadence™ implantation was tolerated VT